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Case Report

Successful treatment with nab-paclitaxel after hypersensitivity reaction to paclitaxel and docetaxel☆

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Introduction

The standard treatment for patients with ovarian cancer is the combination of platinum and taxane chemotherapy. However, one of the limitations in using paclitaxel and docetaxel is the hypersensitivity reaction (HSR) that patients' experience. Despite pretreatment during initial infusion severe HSR could occur in 2% of the cases. For patients who develop HSR to paclitaxel there is the danger of cross reactivity with docetaxel and has been reported to be as high as 90% as previously published by Dizon et al. (2006).

The etiology of HSR to paclitaxel and docetaxel is poorly understood. It is believed that the solvent Cremophor EL in paclitaxel, and polysorbate80 in docetaxel may be the cause of HSR. Yet, others have demonstrated that HSR maybe secondary to the direct effect of the taxane itself (Essayan et al., 1996) and not the diluents. There have been several reports that nab-paclitaxel can be safely administered after HSR to paclitaxel (Fader and Rose, 2009) but no reported cases of safely administering nab-paclitaxel after HSR to both paclitaxel and docetaxel.

Case report

This is a 60 year old female who presented with abdominal pain and bloating. A CT scan of abdomen and pelvis in 7/2012 showed ascites, 16 cm complex pelvic mass and omental caking. In 9/2012 she was brought to the operating room for tumor debulking, however intraoperatively she became hypotensive and unstable. Only partial omentectomy could be performed and pathology was consistent with serous carcinoma from ovarian primary.

In 9/2012 she received her first cycle of neoadjuvant carboplatin and paclitaxel. She was premedicated (diphenhydramine 50 mg IV, decadron 20 mg IV and famotidine 20 mg IV) prior to starting paclitaxel (175 mg/m^2/3 h) however, 20 min into the infusion she developed urticarial rash and went to respiratory distress with O_2 saturation of 60% on room air. Infusion was discontinued and she received epinephrine and solumedrol with resolution of symptoms and improvement of vital signs. In 10/2012, she received second cycle of carboplatin (5 AUC) without complications and received docetaxel (75 mg/m^2/1 h) as a replacement for paclitaxel. Despite receiving premedication with decadron 20 mg IV and diphenhydramine 25 mg IV, 10 min into the docetaxel infusion she developed dyspnea and hypoxia. Given second episode of HSR with taxane, we administered liposomal doxorubicin on her third cycle of chemotherapy; but unfortunately she also developed HSR to liposomal doxorubicin.

Given her history of HSR to paclitaxel, docetaxel and liposomal doxorubicin, a decision was made to try nab-paclitaxel. In 11/2012 she received her first cycle of nab-paclitaxel (100 mg/m^2/30 min) in an outpatient setting after receiving dexamethasone 20 mg IV and diphenhydramine 50 mg IV without evidence of HSR. She subsequently received two more cycles without complications. After six cycles of carboplatin and three cycles of nab-paclitaxel, a repeat CT showed good tumor response and she had an uncomplicated optimal, interval tumor debulking.

Discussion

First line treatment for ovarian cancer is the combination of platinum and taxane however, one of the treatment limiting factors of taxanes is the high risk of developing HSR which could be life threatening. The inability to tolerate and receive a taxane regimen can have a negative impact in patient's overall survival. Acknowledging the clinical importance of taxanes, this patient elected to undergo another attempt at receiving a taxane after developing HSR to paclitaxel and

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docetaxel. It is unclear why she would have had a reaction to paclitaxel and docetaxel but not nab-paclitaxel. One may conclude that patients' who react to both paclitaxel and docetaxel may be reacting to the taxane component and not to the diluents and therefore at higher risk of mounting a HSR if treated with nab-paclitaxel. To the contrary, this patient was safely treated with nab-paclitaxel after HSR to both paclitaxel and docetaxel which supports the idea that the diluents may be the true cause of HSR and not the taxane component itself.

Given the known risk of HSR to paclitaxel and docetaxel, premedication has become the standard of care. However several case reports have been published on the failure of preventing HSR to paclitaxel despite premedication (Del Priore et al., 1995). Options after HSR to paclitaxel include desensitization or changing the chemotherapy regimen. Many physicians would choose to change the paclitaxel to docetaxel and allow for continued treatment with a taxane. Only case reports have been published of physicians directly changing to nab-paclitaxel (Fader and Rose, 2009) after HSR to paclitaxel and to our knowledge there have been no reported cases of using nab-paclitaxel after HSR to both paclitaxel and docetaxel.

Nab-paclitaxel may not be the treatment of choice after HSR to paclitaxel as little is known about its effectiveness in ovarian cancer. Nab-paclitaxel is a solvent free, albumin bound paclitaxel that is administered without requiring premedication. Although there are no studies that show equivalent effectiveness of nab-paclitaxel to paclitaxel or docetaxel, there is a phase II GOG trial (Coleman et al., 2011) that has demonstrated that nab-paclitaxel may be effective in patients who have recurrent or persistent platinum resistant ovarian cancer.

We present the first case report of successfully re-treating a patient with severe HSR to paclitaxel and docetaxel with Nab-paclitaxel in order to continue treating a patient with a taxane.

Conflict of interest

All authors have no conflict of interest to disclose.

References

Coleman, R.L., Brady, W.E., McMeekin, S., Rose, P.G., Soper, J.T., et al., 2011. A phase II evaluation of nanoparticle, albumin-bound nab-paclitaxel in the treatment of recurrent or persistent platinum-resistant ovarian, fallopian tube, or primary peritoneal cancer: a Gynecologic Oncology Group study. Gynecol. Oncol. 122, 111–115.

Del Priore, G., Smith, P., Warshal, D.P., Dubeshter, B., Angel, C., 1995. Paclitaxel associated hypersensitivity reaction despite high-dose steroids and prolonged infusions. Gynecol. Oncol. 11, 2456–2458.

Dizon, D.S., Schwartz, J., Rojan, A., Miller, J., et al., 2006. Cross-sensitivity between paclitaxel and docetaxel in women's cancers programs. Gynecol. Oncol. 100, 149–151.

Essayan, D.M., Kagey-Sobatka, A., Colarusso, P.J., Lichtenstein, L.M., et al., 1996. Successful parenteral desensitization to paclitaxel. J. Allergy Clin. Immunol. 97, 42–46.

Fader, A.N., Rose, P.G., 2009. Abraxane for the treatment of gynecologic cancer patients with severe hypersensitivity reactions to paclitaxel. Int. J. Gynecol. Cancer 19, 1281–1283.